Study Guide for Response Analyses

Summary of this Module

1. Response analyses are designed to identify to how listed resources are likely to respond to being exposed to potential stressors and subsidies produced directly or indirectly by federal Actions and any interrelated and interdependent activities.
2. Response analyses involve three basic steps: (a) gather evidence of the potential responses of listed resources (or surrogates) to an Action's stressors, subsidies, or both; (b) critically appraise that evidence to identify the responses that have the best support in the available evidence; and (c) identify the responses that are most likely given the prior condition of the listed resources and the particular circumstances surrounding a federal Action.
3. Response analyses based on individual organisms include an additional step: connecting the responses of those individuals (given their prior condition) to variables that are relevant demographically.
4. Habitat-based response analyses begin by identifying the probable responses of a species' habitat to stressors or subsidies produced directly or indirectly by federal Actions, then they identify the response of listed species to those changed habitat conditions.
5. Response analyses for listed species (as opposed to analyses for designated critical habitat) begin with the responses of the individuals that would be exposed to an Action's direct or indirect effects on the environment, which requires an accurate assessment of the antecedent conditions of those individuals.

1.0 Introduction

Exposure analyses identify how many individuals of which species are likely to co-occur with an Action's effects and the details of that co-occurrence — what they would be exposed to and how that exposure would vary with space and over time. Given that exposure, it is important to know how those individuals are likely to respond to that exposure. Are they likely to die? Delay reproduction? Produce fewer young or seeds? Grow slower and take longer to mature? Stop feeding? Abandon their territory?

Similar questions follow any exposure analyses for critical habitat. Is the forage base likely to decline in all or a portion of the designated area? Would the area become overgrown? Would tem-

peratures increase (or decrease)? Would the soil or substrate acidify? Would the incidence of fire increase or decrease? Response analyses answer these questions. Response analyses determine how listed resources are likely to respond after being exposed to an Action's effects on the environment or directly on listed species themselves. For habitat-based assessments, response analyses are where we establish relationships between habitat change and a listed species' response to that change. In short, response analyses help translate exposure into risk.

The idea behind response analyses is not new: Service consulting biologists already include the essence of response analyses in biological opinions. However, like exposure and risk analyses, response analyses lend structure to consultations that allows the Services to involve Action Agencies (and any applicant) in the process of assessing the effects of their actions on listed resources. Involving Action Agencies in the process of establishing how listed resources are likely to respond upon being exposed to their Action's effects is an important step in providing transparency to interagency consultations and will help them understand the basis for the Service conclusions.

The conclusions of our response analyses should be driven primarily by the evidence available from published sources (journal articles, conference proceedings, etc.), unpublished sources (reports produced by government agencies, consultancies, institutes, and environmental organizations; doctoral dissertations; and master's theses), and any data that Service biologists may have collected or be able to access. Although some of this evidence will need to be interpreted, most of that interpretation should occur as part of Service risk analyses. As a result, this step of our assessments will be driven by the questions we ask, questions the action agency or applicant ask, the quality of our search strategies, and the information we extract from search results.

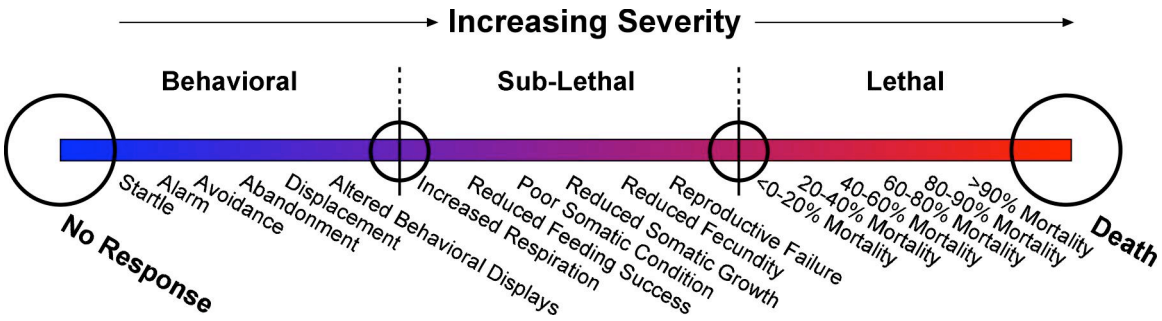
2.0 What Does the "Response" in "Response Analysis" Mean?

Following the analytical framework for consultations, we begin an assessment by subdividing proposed Actions into component parts (which we have called "deconstructing the action") to increase our ability to detect the various pathways and mechanisms by which Actions affect the natural environment. Then we identify the physical, chemical, and biotic effects of those component parts, following them as they move from their source(s) through landscapes, watersheds, coasts, oceans, and the atmosphere over time.

Armed with that information, we then work with an Action Agency and applicant (if any) to identify the threatened or endangered species and designated critical habitat are likely to co-occur with an Action's direct or indirect effects on the environment (which we have called "exposure analyses"). In addition to determining which listed resources are likely to co-occur with an Action's effects, exposure analyses identify where and when the exposure is likely to occur, how frequently the exposure would occur, how long the exposure would occur, and the intensity of any exposure in terms of it severity (for example, destruction of an organism's habitat instead of habitat modification), concentration (for sediments, chemicals, pathogens, etc.), received levels (for noise and other audible disturbance), or similar units of measure.

Response analyses identify how listed resources are likely to respond or react upon being exposed to an Action's effects. In this case, "responses" mean the physical, behavioral, and physiological reactions of individual, listed species when they are exposed to an Action's effects. It includes

Figure 1. An illustration of the range of animal responses to physical, chemical, or biotic stressors. The mortality values in the right end of the spectrum reflect phenomena that are known to be lethal, but only in a portion of cases



individuals that are wounded or die upon exposure; physiological responses like reduced fecundity or increased spontaneous abortion rates that are evidence of "harm" or "harassment"; behavioral responses like abandoning a territory or site that might also be evidence of "harm" or "harassment"; physiological responses that are evidence of "stress" that may have immediate effect on the individuals that have been exposed, chronic effect on the individuals' fitness, or may simply magnify the individuals organisms' responses to future stressors. Response analyses, then, consist of any acute, chronic, or latent reactions in individual members of listed species that are likely to be exposed to an Action's effects.

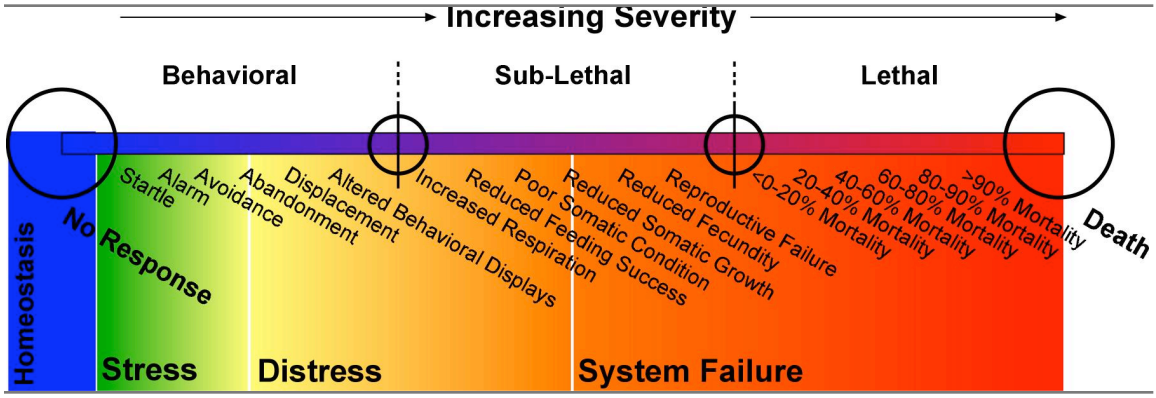
2.1 The Potential Responses of Animals to Human Activities

Animals responses to human activities will generally fall into four categories: no response, physical responses, behavioral responses, and physiological responses (see Figure 1).

1. *No Responses* means no apparent or observed response and encompasses an animal's normal behavioral repertoire. It is important to remember that we should interpret evidence that suggests species did not respond to human activity cautiously: we should distinguish between "no response" and "no apparent response." It will often be impossible for human observers to distinguish between an animal's normal range of responses and its responses to human stimuli.

2. *Physical Responses* encompass the entire range of traumas to the bodies, organs, or tissues of animals caused by natural and anthropogenic stimuli. Physical responses will usually result from physical interactions between an Action's effects and individual animals: capturing an animal in a net, wounding it with a trap, injuring it with electroshocks, shooting it. The most serious of these responses might immediately kill individual animals. Less serious responses might injure the animals or represent traumas that might not kill an animal, but might impair the animal's ability to defend its territory, forage, migrate, reproduce, or rear its young successfully.

Figure 2. An illustration of the relationship between an animal's overt responses and the animal's internal state. As in Figure 1, the mortality values in the right end of the spectrum reflect phenomena that are known to be lethal, but only in a portion of cases.



Physical responses can be acute or chronic. However, unlike other responses, physical responses usually do not involve habituation and often are not reversible.

3. *Behavioral Responses* encompass all behavioral reactions and responses to natural and anthropogenic stimuli. Some of these responses will be reflex responses that an animal would exhibit regardless of the stimulus (for example, an opossum passing out or a vulture "purging" when startled). Some of these responses (such as alert responses or some avoidance) reflect an animal's awareness — a bald eagle that is aware of a human presence, but the human is still to far away to cause the eagle to flush — rather than adverse reactions to a stimulus (we would not detect any differences in pulse rates, respiration rates, energy charges, or hormones that would indicate the animal was stressed in any way). Behavioral responses can be acute or chronic and can often be mitigated by habituation, which would cause an animal to become less sensitive to a stimulus (that is, the animal's response would move toward to left — toward no response — of the scale in Figure 1).

4. P*hysiological Responses* encompass the full range of internal changes in body function and chemistry that are associated with "stress." Almost all of these responses are "sublethal" (acute or chronic) responses that affect animal by changing their physiology. Physiological responses include increased production of "stress" hormones like epinephrine and norepinephrine (which are responsible for "fight or flight" responses that increase heart rates and blood flow to muscles) and corticosteroids (which can increase an animal's metabolic rate).

   Some physiological responses, such as production of stress hormones like glucocorticosteroids, will increase an animal's energy demand above its basal energy requirements (called "allostatic load"). These increases in energy demand can result from an animal's daily and seasonal routine, particularly when combined with the extra energy animal's need to migrate, molt, breed, etc. When an animal's physiological responses to an Action's direct or indirect effects dramatically increase the animal's energy demands (a con-

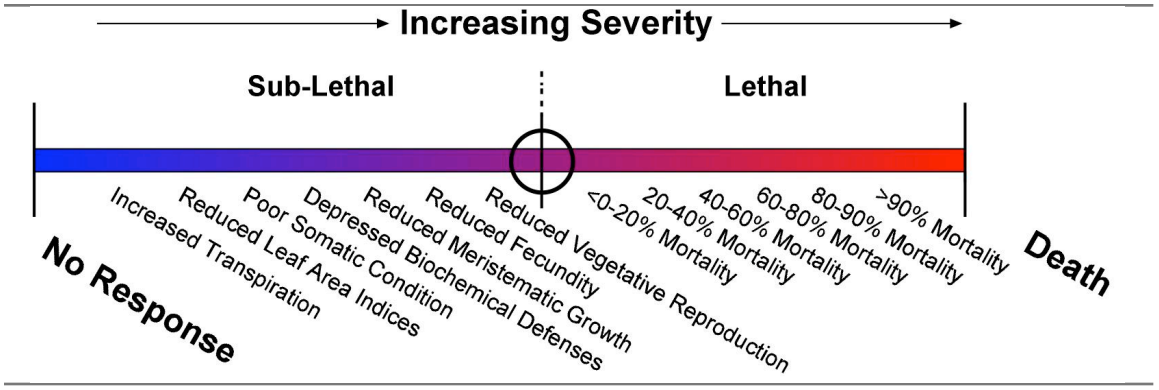
dition called "allostatic overload"), the animal can experience serious pathologies like decreased immune function, stunted growth, and infertility.

When an animal's energy demands exceed its energy income (a condition called "Type 1 allostatic overload"), the animal may enter a "survival mode" or "emergency life history state" that allows the animal to decrease its energy demand and regain positive energy balance. When the stressors responsible for the increased energy demand abate, the animal can return to physiological and energetic states characteristic of a normal life cycle. For example: breeding birds rely on the increased availability of food in the spring to raise their young. If inclement weather increases their homeostatic costs at the same time the birds face the additional demands of breeding and rearing young and also reduces the supply of food the birds need to sustain increased energy demand (increased allostatic load) associated with breeding, then individual birds will face negative energy balances, loss of body mass, and may suppress reproduction. Other physiological responses appear as reductions in the size, number, and viability of an animal's eggs, or increases in the number of miscarriages.

Animals will often try to mitigate some physiological responses through behavior and we will often use these behavioral responses as evidence of physiological responses or "stress": increases in an animal's respiration (for example, increased panting in terrestrial vertebrates or increased surfacing rates in aquatic mammals), reductions in an animal's foraging activity and foraging success, and reduced body condition and reduced growth rates (which can result from reduced foraging success, but can also indicate physiological stress). Physiological responses can be acute or chronic, lethal or sub-lethal. Similarly, some physiological responses are reversible (generally the responses enveloped by the "distress" zone in Figure 2).

Figure 1 illustrates the range of responses that would be apparent or observable by a human observer, but — absent visual symptoms like depleted somatic condition, disorientation, or disease — these responses provide no information on the internal state of the animal. An animal's internal state, particularly physiological and biochemical indicators of stress, is a more sensitive and robust indicator of an animal's response to stressors in its environment (Figure 2). As the left side of Figure 2 illustrates, we might conclude that an animal has had "no response" to an Action's effects when, in fact, the animal is experiencing internal stress (Fair and Becker 2000, Gill *et al*. 2001). For that reason, when we conclude that an animal has had "no response" to an Action's effects, we have some risk of reaching a false conclusion. That risk increases when we rely on studies or evidence that have not measured the internal state of study subjects (see Baker and Johanos 2002 and Krausman *et al*. 1998 for examples of studies that examined the effects of human activity on the internal states of listed animals). The risk of falsely concluding "no response" is highest with behavioral and sub-lethal responses which require humans to interpret an animal's behavior or interpret data on an animal's health (which may also result from differences between individuals in a population or environmental change that is unrelated to an Action's effects).

Figure 3. An illustration of the range of plant responses to physical, chemical, or biotic stressors. The mortality values in the right end of the spectrum reflect phenomena that are known to be lethal (and, in plants, can include grazers or disease), but only in a portion of cases.



2.2 The Potential Responses of Plants to Human Activities

In plants, responses will generally fall into four categories: no response, sub-lethal responses, and lethal responses (see Figure 3).

1. *No Responses* usually means "no apparent" or "no observed" response and encompasses a plant's normal growth rate, phenology (annual cycle of seedling emergence, floral development, seed formation, etc.), visual appearance, or fecundity. Because it will often be impossible for human observers to distinguish between a plant's normal range of responses and its responses to a human stimulus, carefully review the experimental designs of studies that conclude "no response" to be certain that the study could have reached that conclusion.

2. *Physical Responses* encompass the entire range of traumas to the bodies, organs, or tissues of animals caused by natural and anthropogenic stimuli. Physical responses will usually result from the direct effects of Actions on individual animals. The most serious of these responses would immediately kill individual animals. Less serious responses would injure the animals or represent traumas that might not kill an animal, but impair the animal's ability to defend its territory, forage, migrate, reproduce, or rear its young successfully.

   Physical responses can be acute or chronic. However, unlike other responses, physical responses usually do not involve habituation and often are not reversible.

3. *Physiological Responses* encompass the full range of internal, chemical changes that generally indicate "stress" in plants. Like the physiological responses of animals, plants also exhibit sub-lethal responses to stress through mechanisms like increased transpiration or other signs of water stress, reductions in the area of vegetative organs, poor condition, reduced growth rates (which indicate water or biochemical stress), reductions in the number or size of seeds and reduced vegetative reproduction (in some species, reduced reproductive success or sexual reproduction can lead to increased vegetative reproduction). Sub-lethal responses can be acute or chronic.

3.0 Response Analyses for Listed Species

Thus far, we have identified the different ways that species can potentially respond to stressors and subsidies produced directly or indirectly by an Action. **Step one** of response analyses, which consists of identifying an organism's physical, behavioral, and physiological potential responses, requires consulting biologists to collect and analyze some data, this part of response analyses is relatively simple. **Step two** of response analyses

is more challenging because it requires consulting biologists to convert any physical, behavioral, and physiological responses they identify in Step 1 into specific demographic or life-history variables. By completing this conversion, consulting biologists lay a foundation for the risk analyses that lie at the heart of any consultation.

Any assessment of the risks human activities or natural phenomena will begin by assessing changes in one or more of the following demographic (or life-history) variables: changes in the number of individuals in a population, changes in their survival (or mortality), growth rates of individuals (increases in body size), fecundity, maternity, and dispersal (immigration and emigration). These variables have been shown to have the most consistent relationships with a population's rates of growth or decline (usually expressed as a population's continuous rate of increase, *r*, or the population's finite rate of increase, λ or lambda) and, consequently, the most consistent relationships between a population's chances of surviving or risk of extinction (Caswell 2001, Dennis *et al*. 1991, Heppell *et al*. 2000, Morris and Doak 2002, Oli and Dobson 2003, Rae and Ebert 2002, Ratsirarson *et al*. 1996, Ratner *et al*. 1997, Reed *et al*. 1998, Stearns 1992). In greater detail, the variables are:

1. **Number of individuals** of a particular age or stage in a particular time interval (usually denoted n_x). This demographic term captures the effects of Actions that kill individuals and, therefore, reduce the number of individuals of a particular age or stage. It also captures other important changes in a population's fitness: for example, reducing maternity rates in a population will reduce the number of young-of-the-year individuals in the population; as this smaller cohort ages and becomes reproductive, the number of adults in the population will be smaller (without immigration).

   When considering the changes in the number of individuals in a population, our response analyses should always try to distinguish between the number of individuals in different ages or stages that would exhibit a response. Actions that kill eggs have different consequences for populations than actions that kill juveniles, sub-adults, breeding adults, or post-reproductive adults. Quantitative studies of the ecology of species that are long-lived, become sexually mature late in life, and produce small numbers of young have demonstrated that adult survival (usually adult females) often has the greatest effect on population's rates of growth or decline (for examples, see Caswell 2001, Forys and Humphrey 1999, Heppell *et al*. 2000, Marmontel *et al*. 1997, Oli and Dobson 2003, Wiegand *et al*. 1998, Wielgus *et al*. 2001, Wisdom and Mills 1997).

2. **Number** of individuals of a particular age or stage **that survive** during a time interval (usually denoted S_x). This demographic term and its counterpart, the number of individuals that die during a time interval, also captures the effects of Actions that kill individuals

and, therefore, reduce the number of individuals of a particular age or stage. This demographic variable differs from the preceding variable because it addresses the survival (or its substitute, mortality) of individuals rather than there numbers. Using this variable (or its substitute, mortality), we can capture actions that have delayed or latent effects as well as immediate effects on population numbers. Like the previous note, this demographic term also captures other important changes in a population's fitness: for example, reducing the survival of adult females in a population will reduce the population's maternity rates, which will reduce the number of young-of-the-year individuals in the population's next generation, and so on.

With this demographic variable, our response analyses should distinguish between individuals of different ages or stages. Actions that reduce the survival of eggs have different consequences for populations than actions that reduce the survival of juveniles, subadults, breeding adults, or post-reproductive adults.

3. **Growth rates** of individuals plants or animals of a particular age or stage over a particular interval of time (usually denoted g_x[1]). This is one of the most important demographic variables for our risk analyses because of its relationship with other demographic variables. For example, reducing an individual's growth rate can cause the individual to become sexually mature later than other individuals in the same population; it can cause the individual to have a smaller body size when it finally matures; it can cause animals to have smaller numbers of eggs, or smaller eggs; or increase the time interval between repeated spawning (Stearns 1993).

   Changes in growth rates are commonly associated with habitat destruction or modification which reduces the amount, quality, or availability of food or prey in animals. As discussed previously, these kinds of phenomena can push an animal into allostatic overload (increasing an animal's energy demands while reducing the energy available to the animal) which will often reduce the animal's rate of growth (McEwen and Wiegand 2003, Monaghan *et al*. 1992, Oro *et al*. 2004).

Figure 4. A generic illustration of a population's response to habitat change

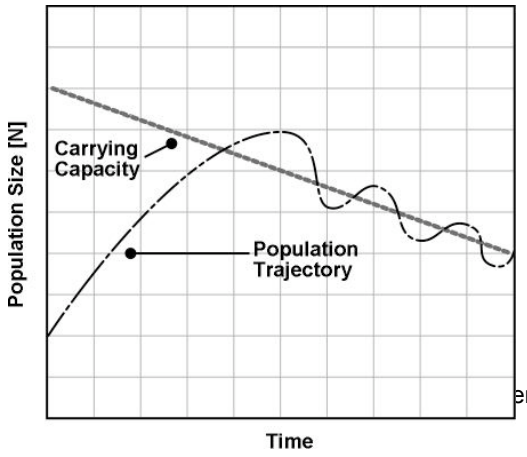
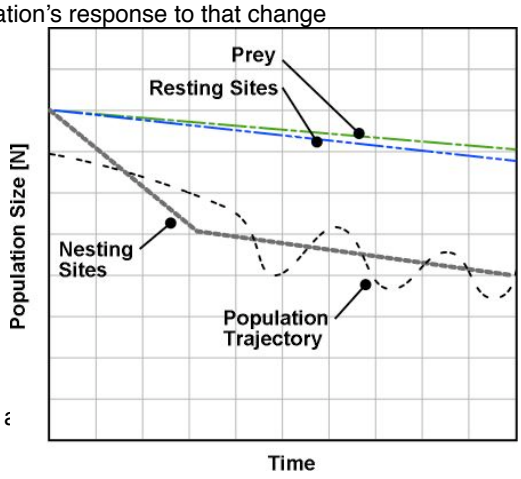


Figure 5. A more accurate illustration of the general relationship between "habitat" change and a population's response to that change



ennis et a

4. Annual **fecundity**— or the number of eggs or seeds — produced by an adult female of a particular age or stage (usually denoted f_x) or increasing the variance in a population's fecundity rates. Here, we distinguish between fecundity and maternity because some adult plants and animals will not dedicate energy to producing eggs or will produce fewer or smaller eggs when they are under stress, while others will produce the same number of eggs, but a greater percentage of those eggs will not be viable (Stearns 1993). Fecundity captures the former response, maternity captures the latter response.

   Like reductions in growth, changes in fecundity are commonly associated with habitat destruction or modification which reduces the amount, quality, or availability of food or prey in animals. In plants, it can be associated with extended drought cycles or flooding, or changes in the availability of soil nutrients. In both plants and animals, reductions in fecundity can be caused by disease or infestation. As discussed previously, reductions in fecundity can be a symptom of allostatic overload (increasing an animal's energy demands while reducing the amount of energy available to the animal, usually by reducing its forage or prey base). For mammals that mature early and have high maternity rates, Oli and Dobson (2003) demonstrated that changes in fecundity was second only to the age of sexual maturity in determining population growth rates (λ).

5. Annual **maternity** — or the number of eggs that survive to hatch, the number of fetuses that are born, or the number of seeds the germinate — for adult females of a particular age or stage (usually denoted m_x). As discussed in the previous note, we distinguish between fecundity and maternity because some adult plants and animals will not dedicate energy to producing eggs or will produce fewer or smaller eggs when they are under stress, while others will produce the same number of eggs, but a greater percentage of those eggs will not be viable (Stearns 1993). Maternity captures the latter response. Maternity also captures more subtle responses: for example, maternity can capture changes in the age or sexual maturity, reductions in the number of second- or third-year adults that given birth, or increases in the number of sub-adults that given birth.

   Like reductions in growth, changes in maternity are commonly associated with habitat destruction or modification which reduces the amount, quality, or availability of food or prey in animals. Chronic stress and its physiological consequences can also reduce an animal's maternity. In plants, it can be associated with extended drought cycles or flooding, or changes in the availability of soil nutrients. In both plants and animals, reductions in fecundity can be caused by disease or infestation. As discussed previously, reductions in fecundity can be a symptom of allostatic overload (increasing an animal's energy demands while reducing the energy available to the animal).

6. **Number** of individuals of a particular age or stage **that immigrate** into a population (usually denoted I_x). Immigration and immigration rates can be critical to the survival of some "open" populations (populations or metapopulations that are connected by dispersing individuals). Immigrants can help prevent inbreeding depression, it can allow individuals to recolonize an area that has been extirpated, it can "rescue" subpopulations that have declined and have become non-viable, or it can allow subpopulations with negative growth rates ("sink" populations) to persist.

If our exposure analyses establish that the population in the Action Area is a "sink" population, it will be particularly important for us to identify any reductions in the number of individuals that immigrate into the population or changes in the age, stage, or reproductive condition of the immigrants.

7. **Number** of individuals of a particular age or stage **that emigrate** from a population (usually denoted E_x). Like immigration, emigration can also be critical to the survival of some "open" populations (populations or metapopulations that are connected by dispersing individuals). Without emigrants, there are no immigrants to help prevent inbreeding depression, recolonize an area that has been extirpated, "rescue" subpopulations that have declined and have become non-viable.

8. If our exposure analyses establish that the population in the Action Area is a "source" population, it will be particularly important for us to identify any reductions in the number of individuals that emigrate from the population or changes in the age, stage, or reproductive condition of the emigrants.

Although most of our risk assessments will be qualitative, the relationships between these variables, population growth rates (or rates of decline), and a species' risks of extinction (or chances of persistence) will remain the same. In addition to these demographic variables, we will also need to retain any information on changes in behavior, because of relationships between animal behavior and the survival of their populations in the wild.

4.0 Response Analyses for Habitat-Based Assessments

Consulting biologists commonly use habitat as a proxy for species. In fact, habitat-based assessments are the most common approach consulting biologists use in consultations. One assumption underlying habitat-based assessments is that the loss and fragmentation of habitat would reduce the size of a population of listed resources (Figure 4). Ecologically, it would be more correct to think of "habitat" as a generic term for the physical, chemical, and biotic resources species need to complete all or portions of their life cycles (Figure 5). That is, Actions do not affect "habitat" they affect the physical, chemical, or biotic resources that individual animals (or plants) require; individual animals respond to changes in those resources (Figure 5).

Habitat-based analyses rely on the relationship between individual animals and the resources they require, but we often only imply those relationships rather than being specific about them. For example, in the situation illustrated in Figure 5, where an Action's effects reduce the number of nesting sites available to a species in a particular area, the individuals are likely to respond with reduced fecundity, maternity, and perhaps reductions in the number of adults in a population as they engage in lethal contests over a nest site. Depending on the species, the population could also experience some emigration as unsuccessful adults leave an area in search of suitable nesting sites. However, adults that successfully find nest sites in an area affected by an Action should reproduce successfully because the Action does not affect their prey base.

The same would not be true with Actions that affect an animal's prey base but not the number of nest sites. Reductions in a species' prey base would affect all members of a population, regardless of their gender, age, or stage. Depending on the magnitude of the reduction, the change in the

population's prey base would reduce individual growth rates, the fecundity or maternity of adults occupying the site, reduce neonate and juvenile survival (or increase their mortality) and reduce adult survival rates.

When we conduct habitat-based jeopardy analyses, in which "habitat modification" or "habitat destruction" are how an Action has demographic effect on individual members or populations of listed species, we need to specify the nature of the habitat change and connect that change to the species' demography. That is, our references to "habitat modification" or "habitat destruction" are generic substitutes for more specific phenomena that have specific demographic consequences for listed species, including disruption or elimination of an organism's prey base, alteration of the cover an organism needs to reduce its risk of predation, alteration of the physical structure an organism uses for reproduction, etcetera. In these instances, we should specify the nature of the habitat change rather than conduct our assessment based on generic "habitat modification" or "habitat destruction.

For example, destroying an organism's prey base and eliminating the cover an organism needs to reduce its risk of predation are both forms of "habitat destruction or modification." However, both of them affect species through different mechanisms and have completely different demographic consequences for species. Destroying an organism's prey base — either by reducing the quantity of prey, its quality, or its availability — increases competition for the remaining prey, may reduce the fertility of adult females, increase the number of live births in adult females, reduces the growth rates of individuals exposed, and tend to have disproportionate affects on younger, smaller, or subordinate individuals. Eliminating the cover an organism needs to reduce its risk of predation increases the predation risks of individual organisms and, depending on the organism, will affect the survival of specific age classes or all age classes equally.

As a result, habitat-based response analyses require us to make an additional step to connect exposure to potential stressors and demographic responses of listed species. Our knowledge of the demographic responses any habitat changes are likely to elicit specific demographic responses in the individuals that occupy the habitat should determine whether we conclude that a habitat change is likely to "adversely affect" listed species. If we cannot build compelling arguments that a habitat change is likely to result in adverse, demographic responses, then we should reconsider any conclusions we might have reached during a consultation. In response analyses we conduct with habitat-based assessments, our "data" are the exposure of specific elements of a species' habitat to stressors associated with elements of an Action, our first conclusion is that those elements of the habitat would change in a particular direction in response to that exposure. Our secondary conclusion is that the individuals occupying the habitat(s) would experience particular demographic responses. As with the earlier steps, we must provide warrants and backing for our habitat-based response analyses. Our backing would generally consist of our studies of patterns of responses in (a) habitats of other populations of the same species, (b) habitats of other species, (c) ecological theory, and (d) computer simulations and modeling exercises.

With habitat-based assessments, our response analyses should establish causal relationships between potential stressor(s), changes in one or more variables in a species' habitat, and a species' response to those changes. In particular, our response analyses for habitat-based assessments should establish that exposure pathways — or the route(s) that potential stressors take from their

source to listed species or critical habitat —are complete. For habitat-based assessments, this criterion will require us to establish a complete connection from an Action, through a species' habitat, to the species' response to the changes in habitat (and support our assertion with evidence supported by literature) to establish a causal relationship between an Action and a species' response. If exposure pathways are not complete, then potential stressors associated with an Action do not reach listed species or critical habitat and, therefore, cannot cause a response. Similarly, our assessments also should establish that (a) the exposure would precede the response; (b) the relationship is biologically plausible based on current understanding of physical, chemical, and biotic processes and mechanisms; (c) the association between potential stressors and a response has been observed consistently in different studies and in different populations.

Finally, populations will generally respond to changes in the feature of their habitat that is the most limiting. The situation illustrated in Figure 5, for example, would have different consequences if the population size was not limited by the quality and quantity of prey, nesting sites, or resting sites; if none of these three habitat features limits the number of individuals an area can sustain, reducing one or more of these three features might not cause the population to respond. Habitat-based assessments assume that particular resources would impose limits on the number of individual animals an area could sustain at a particular level of health. That assumption fails if a population would not be limited by the quantity, quality, or availability of resources as a result of the action. As a result, response analyses should establish that an Action's effects are likely to change one or more features of a species' habitat in ways that can be expected to create new limits on the size of a population or exacerbate limits that already exist.

5.0 Response Analyses for Designated Critical Habitat

Our response analyses for critical habitat will follow the general pattern for habitat-based assessments: we will need to identify the actual resources that are likely to be affected by a proposed action because different resources have different implications for how listed species are likely to respond to an Action's effects. Unlike habitat-based assessments, response analyses for critical habitat should establish relation-ships between an Action's direct and indirect effects and the constituent elements the Services included in the critical habitat designation. In particular, response analyses for critical habitat need to establish relationships between the overall value of the habitat for the conservation of listed species and an Action's effects on specific constituent elements (recognizing that implicit constituent elements would also include spatial variables like the area of critical habitat, its spatial pattern, and connectivity).

6.0 Things to Keep in Mind When You Conduct Response Analyses

1. **Response analyses allow you to translate exposures into risk.** Response analyses provide structured answers to questions about what is likely to happen to threatened and endangered species, designated critical habitat, or both when they are exposed to an Action's effects on the environment. Any investment you make in structuring your response analyses carefully makes the conclusions of your consultation more robust.

2. **Response analyses should be driven by the available evidence**. Although this will require you to gather evidence and evaluate any evidence you gather for reliability and relevance, these analyses should be as objective as possible. Treating response analyses as process of answering a series of neutral questions using the available evidence will help achieve this outcome.

3. **Tailor your response analyses to the probable exposure**. Response analyses should be specific to the Action's effects as they will occur in space and over time, recognizing that the intensity of those effects will often attenuate over space and time. As we prepare our response analyses, we need to remember that potential stressors — like sediment loading, some biocides, and sounds — can become less intense with increasing distance from a source and they can also become less intense as time passes, even close to a source. For example, a prairie-fringed orchid (*Platanthera* spp.) that is exposed to an herbicide near the source of the application will probably exhibit different responses than the same orchid that is affected by wind-borne drift a kilometer from the application. Similarly, sedimentation would elicit different responses from species like Neosho madtom (*Noturus placidus*), loach minnow (*Tiaroga cobitis*), or steelhead smolt (*Oncorhynchus mykiss*) depending on the distance of those species from the source of the sediment; the species response might also change if the species is exposed to the sediment a few days, a week, or a month after the discharge occurred (in the latter case, the species might be exposed to both coarse and fine particles if it was in the area during the sediment discharge, but might be exposed only to fine particles after the coarse particles precipitated from the water column).

4. **Tailor your response analyses to the listed resource**. A plant's or animal's response to a potential stressor will often depend on its age or stage, sex, and its health condition and health history prior to being exposed to the new stressor. For example, seeds in a seed bank will have different responses to light, crown fires than mature, annual plants; glochidia will have different responses to suspended sediment than adult mussels. Your response analyses should be sensitive to the age(s) or stage(s) of the listed resources that would be exposed to an Action's effects; health condition of the individuals; the timing of that exposure in the annual cycle of listed resources (many animals may exhibit responses during one part of their annual cycle and not at others).

5. **Establish causal relationships between exposure and response.** This is an important step with any assessment approach, but is particularly important with habitat-based assessments (see Appendix A for additional recommendations on this step). If you cannot establish a causal relationship between exposure to an Action's effects and a listed resource's response or if you cannot articulate why there is a causal relationship, you should re-evaluate your assessment.

6. **Structure your response analyses to protect against Type II error**. As always, concluding that a listed resource would have no response to an Action's effects when, in fact, the resource would have substantial, adverse responses, could place the species at greater risk of extinction. You increase your chances of Type II error if you fail to identify potential responses, particularly sub-lethal or lethal responses. You can minimize this risk by

verifying the depth and breadth of your personal knowledge through careful searches of the scientific literature and other evidence (see the next section and the Evidence module of this course).

7.0 Dealing with Uncertainty and Unknowns

Despite the wealth of information available, you will encounter some situations where there is no evidence. In those instances, you will need to use surrogates and make inferences about the probable responses of listed resources based on the responses of those surrogates. The best approach to using surrogates is to move up through a species' taxonomic sequence: that is, start with subpopulations, move to populations, then subspecies, species, genus, family, class, and order. You need to remember that as the taxonomic group you may use as a surrogate becomes more removed from the listed species, you increase the risk of coming to a false conclusion.

You can reduce the risk of false conclusions by comparing the life cycles of your surrogates with the listed species (try to compare long-lived species with other long-lived species, compare species with delayed maturity with other species that also have delayed maturity) and comparing the trophic position and ecologies of the two species (try to use top or mid-level predators as surrogates for other top or mid-level predators), and comparing species with analogous vital rates (try to compare slow-growing populations — that is, populations with low rates of increase — with other slow-growing populations). Rather than use a single surrogate, try to use multiple surrogates and make inferences from the entire set.

8.0 Strategies for Working with Action Agencies and Applicants

Although agencies have become accustomed to initiating consultations, then waiting to receive biological opinions, nothing requires the Services to do all of the work of consultation. During consultation, the Services can task Action Agencies, applicants, or both to help the Services conduct response analyses. They (or their consultants) can conduct literature searches, gather other evidence for response analyses (for example, monitoring reports), as well as analyze the results of those searches.

Tradition will be the primary obstacle to using this kind of strategy: Action Agencies have become accustomed to initiating consultation then waiting for a biological opinion. Agencies will assume that once they have submitted a Biological Assessment and the information they must submit to initiate formal consultation (see 50 CFR 402.14(c)), that their responsibilities have ended. It is important to remember that this information is required to initiate a consultation, it is not the consultation itself.

As we have discussed several times in this course, you can help change that tradition by telling Action Agencies and applicants how you plan to conduct your assessment when you begin your consultation. During that discussion, you can establish the roles and responsibilities and expectations. When you respond to an Agency's request to initiate formal consultation, you can also tell them that although they have provided the information that is required to *initiate* formal consultation, additional information or analyses may be required to complete the analyses that are required to complete formal consultation (note that this strategy allows that our regulations only identify

the information required to *begin* a consultation which may be different than the information that is required to *complete* a consultation).

As always, it is important to remain reasonable with your requests and your expectations. You should not ask Action Agencies or applicants to provide you with information or analyses that you already have or that you can access more easily than they can (for example, if the information is generated by Service personnel or by a State agency with Federal aid funds or recover funds, the Services would have more access to that information than most Action Agencies). You should not ask Action Agencies or applicants for analyses they cannot conduct or do not know how to conduct.

When you have complete your response analyses, you should consider providing Action Agencies and applicants with an opportunity to review and comment on your results (this will be more important in controversial consultations). Their review should be limited to substantive issues that can be supported with evidence, such as:

1. our analyses omitted or neglected information or evidence that might lead to different conclusions (with the evidence provided or referenced), or
2. our search strategy omitted or neglected sources of relevant evidence (with the sources provided or referenced).

If you receive comments on response analyses, make certain that your administrative record contains your written response to those comments.

9.0 Documenting Response Analyses

When your response analyses are complete, the administrative record for your consultation should have:

1. Any correspondence between the Services, Action Agencies, and applicants the relate to these analyses;
2. Copies of literature searches you, Action Agencies, or applicants might have conducted to complete these analyses, supported by specific information on the search strategy that was used to gather evidence;
3. Memoranda to the consultation file that identifies the information you used in your analyses, evidence you discounted for your analyses (and an explanation of why), how you analyzed any evidence in your analyses, and the results your analyses produced;
4. Letters, e-mail messages, memoranda, or other documentation of any reviews of your response analyses and how you addressed any comments you received.

Glossary of Terms

Allostasis: maintaining stability through change, as a fundamental process through which organisms actively adjust to both predictable and unpredictable events. Allostasis is a process that supports homeostatis as an organism's life history stage change, its environment changes, or both. Allostasis is distinguished from homeostasis because the former is designed to keep homeostatic mechanisms in balance when an organism's life history changes or its environment changes while homeostasis refers to stability in the physiological systems that are essential for the organism's life.

Allostatic load: refers to the cumulative "costs," usually in terms of energetic demands above those required for homeostasis, of allostasis or an allostatic state. An allostatic load can the result of the daily and seasonal routines organisms have to obtain food and survive combined with the extra energy organisms need to migrate, molt, breed, etc. Within limits, allostatic loads are adaptive responses to seasonal and other demands

Allostatic overload: a physiological state or states that occur when energetic demands (allostatic loads) from unpredictable events in the environment dramatically increase an organism's energetic requirements and which serious pathophysiology can occur. Using the balance between energy input and expenditure as the basis for applying the concept of allostasis, McEwen and Wingfield (2003) proposed two types of allostatic overload.

a. Type 1 allostatic overload occurs when an organism's energy demands exceed its energy income, resulting in activation of the emergency life history state (a survival mode that decreases allostatic loading and regains positive energy balance). When the stressors responsible for the allostatic overload abate, the organism can return to the physiological and energetic states characteristic of its normal life cycle.

   Example: breeding birds rely on the increased availability of food in the spring to raise their young. If inclement weather increases their homeostatic costs at the same time the birds face the additional demands of breeding and rearing young and also reduces the supply of food available to sustain the allostatic loads associated with breeding, then individual birds will face negative energy balances, loss of body mass, and suppression of reproduction.

b. Type 2 allostatic overload begins when there is sufficient or even excess energy consumption accompanied by social conflict and other types of social dysfunction. The latter is the case in human society and certain situations affecting animals in captivity. In all cases, secretion of glucocorticosteroids and activity of other mediators of allostasis such as the autonomic nervous system, CNS neurotransmitters, and inflammatory cytokines wax and wane with allostatic load. If allostatic load is chronically high, then pathologies develop. Type 2 allostatic overload does not trigger an escape response, and can only be counteracted through learning and changes in the social structure.

Allostatic state: refers to altered and sustained activity levels of primary mediators (for example, glucocortisteroids) that integrate an organism's physiology and associated behaviors with changing environments and challenges such as social interactions, weather, disease, predators, pollution,

etc. Allostatic states usually increase an organism's energetic demands above basal levels and, consequently, can be sustained for limited periods of time when supplies or food or stored energy are available. If an organism's allostatic state continues for long periods of tie or becomes independent of adequate energy reserves, then symptoms of allostatic overload appear.

Association: an association exists if two variables appear to be related by a mathematical relationship; that is, a change of one appears to be related to the change in the other. An association is necessary for a causal relationship to exist but an association alone does not establish a causal relationship. Correlation coefficients or risk measures often quantify associations. There are two basic types of associations:

a. **Negative association** (Inverse Relationship): The magnitude of one variable appears to move in the opposite direction of the other associated variable. The correlation coefficient is negative and, if the relationship is causal, higher levels of the risk factor are protective against the outcome.

b. **Positive association** (Direct Relationship): The magnitudes of both variables appear to move together up or down. The correlation coefficient is positive and, if the relationship is causal, higher levels of the risk factor cause more of the outcome.

Critical appraisal: the concepts and methods of critical thinking used to answer the key question "How good (strong) is the evidence for that?" when evaluating evidence for use in the practice of clinical medicine, whether the evidence is from clinical observations, laboratory results, scientific literature, or other sources (after answering the question "What is the evidence for that?").

Critical stimulus level: the stimulus level (current, dose, energy, stress, voltage, etc.) that is just sufficient to cause a response in a specimen tested. If the specimen is subjected to a stimulus level below its critical stimulus level it will not respond. If it is subjected to a stimulus level above its critical stimulus level it will respond

Critical thinking: the disciplined ability and willingness to assess evidence and claims, to seek a breadth of contradicting as well as confirming information, to make objective judgments on the basis of well supported reasons as a guide to belief and action, and to monitor one's thinking while doing so (metacognition). The thinking process that is appropriate for critical thinking depends on the knowledge domain (e.g.: scientific, mathematical, historical, anthropological, economic, philosophical, moral) but the universal criteria are: clarity, accuracy, precision, consistency, relevance, sound empirical evidence, good reasons, depth, breadth and fairness.

Determinant: any factor, whether event, characteristic, or other definable entity, that brings about a change in health condition or other defined characteristic.

Determinant, distal (distant): an established or postulated factor that is remote or far apart in position, time, or resemblance to the outcome of concern, making it difficult to discern or trace the causal pathway. An example is atmospheric contamination with ozone-destroying substances that may increase the risk of skin cancer. Also upstream determinant.

Determinant, proximal (proximate): an established or postulated factor that is nearest in time, distance, or both to an outcome of concern. The causal pathway is clearly defined and allows a confident assertion or the causal linkage between the determinant and the outcome.

Ecological response analysis: a step in the process of characterizing ecological effects that examines three primary elements: (a) the relationship between stressor levels and ecological effects, also called stressor-response analysis; (b) the plausibility of effects occurring as a result of exposure; and (c) linkages between measurable ecological effects and assessment endpoints when the latter cannot be directly measured.

Ecological trap. in an environment that has been altered suddenly by human activities, an organism makes a maladaptive habitat choice based on formerly reliable environmental cues, despite the availability of higher quality habitat. An ecological trap is a specific type of EVOLUTIONARY TRAP.

Effect modifier: a factor that modifies the effect of a putative causal factor under study. For example, age is an effect modifier for many conditions. Effect modification is detected by varying the selected effect measure for the factor under study across levels of another factor.

Latent effects: direct or indirect stimuli that have a delayed response in an individual, population, or species that is exposed to those stimuli (at levels, intensities, frequencies, or durations that would be expected to elicit a response). Latent effects can be positive or negative. For example, a prescribed burn that is conducted in the fall would have latent, beneficial effects on individuals, populations, or species that depends on the post-burn vegetative community.

Latent period: the time between the biologic onset of a disease or condition and the detection of the disease or condition. For example, because we census sea turtles based on counts of adult females, there may be a long latent period before we might detect the effects of juvenile and subadult mortalities on a population's trend. Detection can be clinical, with the appearance of clinical signs, or detection can be sub-clinical, which require positive diagnostic tests.

Lines of evidence: Information derived from different sources or by different techniques that can be used to describe and interpret risk estimates. Unlike the term ``weight of evidence,'' it does not necessarily imply assignment of quantitative weightings to information.

Measurement of response. The assignment of numbers to responses or to response-instances according to the rules of measurement. Measures include amplitude of response, intensity of response, latency of responses, probability of response, rate of response (V).

Shelford's law of tolerance: a law proposed by V.E. Shelford which states that an organism's presence and success depend on the extent to which a complex of conditions is satisfied (for example, the organism's climatic, topographic, and biological requirements). By extension, an organism's absence or failure can be controlled by qualitatively or quantitatively reducing or increasing any one of several factors to approach of exceed the organism's limits of tolerance for that factor.

Stress: the response of a system to a stressor, which may include adaptation or functional disorder. An internal state brought about by a stressor.

Stress ecology: Seyle (1950) provided what has become the classical definition of stress in a treatise that dealt with the concept of stress from the perspective of modern medicine. He defined stress as the sum of all physiological responses by which an animal attempts to maintain or reestablish a normal metabolism in the face of a physical or chemical force.

Brett (1958) redefined stress so that it would encompass non-human animals. He defined stress as "a state produced by an environmental or other factor which extends the adaptive responses of an animal beyond the normal range, or which disturbs the normal functioning to such an extent that, in either case, the chances of survival are significantly reduced." He phrased this definition to make it possible to (1) identify a normal range of responses, (2) quantify stress by measurements of changes in performance from normal levels, and (3) recognize that stress is disadvantageous to the organism affected by it.

More recent definitions of stress either accept and reject Brett's (1958) definition of stress as a state that is detrimental to an organism. Bayne (1975), Odum (1971), Odum et al. (1979), and Lugo (1978) stated that a definition of stress should indicate that it is detrimental to an organism or ecosystem. However, Esch and Hazen (1978), Esch *et al*. (1975), and Seyle (1974) stated that a stressor can have a neutral effect on a stressed system as well as a detrimental one. In his review of the subject, Seyle (1974) indicated that, in the context of human medicine, stress does not always result in reduced viability and distinguished between forms of stress that could result in beneficial effects and those that were detrimental to the human system. He applied the term eustress to phenomena that challenge the human system in a manner that results in development of an adaptive response that enhances human welfare. He applied the term distress to phenomena that initiate processes of system failure characterized by irreversible physiological transformations which lead ultimately to death.

To allow for the existence of stressors that can be beneficial to a system as well as those that are detrimental, Esch *et al*. (1975) defined stress as "the effect of any force which tends to extend any homeostatic or stabilizing process beyond its normal limit, at any level of biological organization." By defining stress in this manner, Esch *et al*. (1975) not only allowed stress to be beneficial, they also expanded the definitions of Brett (1958) and Seyle (1950, 1952, 1956, 1974) to encompass any biological or ecological entity. By including the phrase "homeostatic or stabilizing process," this definition also makes it implicit that ecosystems have stabilizing forces operating within them that are comparable to those operating within organisms.

Bayne (1975) defined stress as "measurable alteration of a physiological (or behavioral, biochemical, or cytological) steady-state which is induced by an environmental change, and which renders the individual (or the population or community) more vulnerable to further environmental change." Bayne (1975) believed that the definition of stress should include a demonstration of "disadvantageous," but recognized that measurement of survival potential is difficult.

Barrett *et al*. (1976) defined stress simply as a perturbation [stressor] applied to a system which is (a) foreign to that system, or (b) which is natural to a system, but applied at an excessive level (e.g., nitrogen, phosphorus, or water). Problems with this definition are that it lacks the concept of "disadvantage" that was introduced in earlier definitions. As Odum *et al*. (1979) indicated, the concept of stress should refer to negative deflections.

Lugo (1978) re-emphasized that an important characteristic of stressed systems is that, in them, energy expenditure increased or potential energy decreased. Both Lugo (1978) and Odum *et al*. (1979) provided discussions which support the conclusion that stress places an organism or system at a disadvantage since a continued increase in energy expenditure is incompatible with survival.

Stress regime: the term ``stress regime'' has been used in at least three distinct ways: (1) to characterize exposure to multiple chemicals or to both chemical and non-chemical stressors (more clearly described as multiple exposure, complex exposure, or exposure to mixtures), (2) as a synonym for exposure that is intended to avoid overemphasis on chemical exposures, and (3) to describe the series of interactions of exposures and effects resulting in secondary exposures, secondary effects and, finally, ultimate effects (also known as *risk cascade* [Lipton et al., 1993], or *causal chain*, *causal network*, or *causal pathway* [Andrewartha and Birch, 1984]).

Stressor: any physical, chemical, or biological entity that can induce an adverse response (synonymous with *agent*). Any condition or situation that causes a system to mobilize its resources and increase its energy expenditure (Seyle 1956, Fitch and Johnson 1977).

Stressor-response analysis: the process of identifying relationships between stressors and responses to those stressors. These analyses determine if (a) the assessment requires point estimates or stressor-response curves; (b) the assessment requires establishment of a “no-effect” level; (c) cumulative distributions would help the assessment; and (d) the analyses would be used as inputs to process models.

Stressor-response profile: the product of characterization of ecological effects in the analysis phase of ecological risk assessment. A stressor-response profile summarizes the data on the effects of a stressor and the relationship of the data to an assessment endpoint.

Susceptibility: having a constitution or temperament that is open, subject, or nonresistant to an agency, influence, intervention, or stimulus.

Threshold dose: the dose above which effects occur.

Threshold phenomena: events or changes that only occur after a certain level of a characteristic is reached (also called a “tipping point”).

Vitality: an abstract property that changes with the moment-to-moment experiences of an organism. An organism’s resistance to disease, level of stress, behavior, success and failure in feeding, frequency of predator attacks, mating, parental care, and habitat choice all induce incremental changes in vitality. By definition the *vitality based mortality* is stochastic and mortality occurs when vitality reaches zero. Mortality can also occur independent of an organism’s vitality through an *accidental based mortality* (Anderson 1992, 2000).

Weight-of-Evidence: the process by which measurement endpoints are related to an assessment endpoint to evaluate whether a significant risk of harm is posed to an ecological entity. The approach is planned and initiated at the problem formulation stage of an ecological risk assessment and results are integrated at the risk characterization stage.

Literature Cited

Adams, S. M. 2003. Establishing causality between environmental stressors and effects on aquatic ecosystems. Human and Ecological Risk Assessment **9**:17-35.

Anderson, J. J. 1992. A vitality-based stochastic model for organism survival. Pages 256-277 *in* D. L. DeAngelis and L.J. Gross, editors. Individual-based models and approaches in ecology. Chapman & Hall;, New York, New York.

Anderson, J. J. 2000. A vitality-based model relating stressors and environmental properties to organism survival. Ecological Monographs **70**:445-470.

Baker, J. D., and T. C. Johanos. 2002. Effects of research handling on the endangered Hawaiian monk seal. Marine Mammal Science **18**:500.

Beyers, D. W. 1998. Causal inference in environmental impact studies. Journal of the North American Benthological Society **17**:367.

Caswell, H. 2001. Matrix population models. Sinauer Publishers, Inc., Sunderland, Massachusetts.

Collier, T. 2003. Forensic ecotoxicology: establishing causality between contaminants and biological effects in field studies. Human and Ecological Risk Assessment **9**:259-266.

Dennis, B., P. L. Mulholland, and J. M. Scott. 1991. Estimation of growth and extinction parameters for endangered species. Ecological Monographs **61**:115-143.

Evans, A.S. 1976. Causation and disease: the Henle-Koch postulates revisited. Yale Journal of Biological Medicine 49: 175-195.

Fair, P. A., and P. R. Becker. 2000. Review of stress in marine mammals. Journal of Aquatic Ecosystem Stress and Recovery 7:335-354.

Forys, E. A., and S. R. Humphrey. 1999. Use of population viability analysis to evaluate management options for the endangered lower keys marsh rabbit. Journal of Wildlife Management 63:251-260.

Gill, J. A., K. Norris, and W. J. Sutherland. 2001. Why behavioral responses may not reflect the population consequences of human disturbance. Biological Conservation 97:265-268.

Heppell, S. S., H. Caswell, and L. B. Crowder. 2000. Life histories and elasticity patterns: Perturbation analysis for species with minimal demographic data. Ecology **81**:654-665.

Hill, A.B. 1965. The environment and disease: association or causation? Proceedings of the Royal Society of Medicine 58: 295-300.

Krausman, P. R., M. C. Wallace, C. L. Hayes, and D. W. DeYoung. 1998. Effects of jet aircraft on mountain sheep. Journal of Wildlife Management **62**:1246-1254.

Marmontel, M., S. R. Humphrey, and T. J. O'Shea. 1997. Population viability analysis of the Florida manatee (*Trichechus manatus latirostris*), 1976-1991. Conservation Biology 11:467-481.

McCauley, R. D., J. Fewtrell, and A. N. Popper. 2003. High intensity anthropogenic sound damages fish ears. The Journal of the Acoustical Society of America **113**:5.

McEwen B.S. and J.C. Wingfield. 2003. The concept of allostasis in biology and biomedicine. Hormones and Behavior 43(1):2-15.

Monaghan, P., J. D. Uttley, and M. D. Burns. 1992. Effect of changes in food availability on reproductive efffort in Arctic terns, *Sterna paradisea*. Ardea **80**:71-81.

Morris, W. F., and D. F. Doak 2002. Quantitative conservation biology. Theory and practice of population viability analysis. Sinauer Associates, Inc. Publishers, Sunderland, Massachusetts.

Norton, S. B., S. B. Cormier, S. M. Cormier, G. W. Suter II, D. Altfater, and B. Counts. 2002a. Determining the causes of impairments in the Little Sci-

oto River, Ohio, USA: Part 2. Characterization of causes. Environmental Toxicology and Chemistry **21**:1125-1137.

Norton, S. B., S. B. Cormier, S. M. Cormier, G. W. Suter II, B. Subramanian, E. Lin, D. Altfater, and B. Counts. 2002b. Determining probable causes of ecological impairment in the Little Scioto River, Ohio, USA: Part 1. listing candidate causes and analyzing evidence. Environmental Toxicology and Chemistry **21**:1112-1124.

Norton, S. B., S. B. Cormier, G. W. Suter II, and S. M. Cormier. 2002c. A methodology for inferring the causes of observed impairments in aquatic ecosystems. Environmental Toxicology and Chemistry **21**:1101.

Norton, S. B., S. M. Cormier, and G. W. Suter II. 2002d. The easiest person to fool. Environmental Toxicology and Chemistry **21**:1099.

Oli, M. K., and F. S. Dobson. 2003. The relative importance of life-history variables to population growth rate in mammals: Cole's prediction revisited. American Naturalist **161**:422-440.

Popper, A. N., R. D. McCauley, and J. Fewtrell. 2002. Impact of anthropogenic sounds on fishes. Journal of the Acoustical Society of America **112**:2431.

Rae, J. G., and T. A. Ebert. 2002. Demography of the endangered fragrant prickly apple cactus, *Harrisia fragrans*. International Journal of Plant Sciences 163:631-640.

Ratner, S., R. Lande, and B. B. Roper. 1997. Population viability analysis of spring chinook salmon in the South Umpqua River, Oregon. Conservation Biology 11:879-889.

Ratsirarson, J., J. A. Silander, Jr., and A. F. Richard. 1996. Conservation and management of a threatened Madagascar palm species, *Neodypsis decaryi*, Jumelle. Conservation Biology 10:40-52.

Reed, J. M., C. S. Elphick, and L. W. Oring. 1998. Life-history and viability analysis of the endangered Hawaiian stilt. Biological Conservation 84:35-45.

Stearns, S. C. 1992. The evolution of life histories. Oxford University Press, New York, New York.

Susser, M.W. 1991. What is a cause and how do we know one? A grammar for pragmatic epidemiology. American Journal of Epidemiology 133: 635-648.

Susser, M. 1995. Judgment and causal inference: criteria in epidemiologic studies. American Journal of Epidemiology 141:701.

Wiegand, T., J. Naves, T. Stephan, and A. Fernandez. 1998. Assessing the risk of extinction for the brown bear (*Ursus arctos*) in the Cordillera Cantabrica, Spain. Ecological Monographs 68:539-570.

Wielgus, R. B., F. Sarrazin, R. Ferriere, and J. Clobert. 2001. Estimating effects of adult male mortality on grizzly bear population growth and persistence using matrix models. Biological Conservation 98:293-303.

Wisdom, M. J., and L. S. Mills. 1997. Sensitivity analysis to guide population recovery: Prairie-chickens as an example. Journal of Wildlife Management 61:302-312.

FURTHER READING

Adams, S. M., W. D. Crumby, M. S. Greeley, Jr., M. G. Ryon, and F. M. Schilling. 1992. Relationships between physiological and fish population responses in a contaminated stream. Environmental Toxicology and Chemistry 11:1549.

Anthony, L. L., and D. T. Blumstein. 2000. Integrating behaviour into wildlife conservation: The multiple ways that behavior can reduce N_e. Biological Conservation **95**:303-315.

Auerbach, S. I. 1981. Ecosystem response to stress: a review of concepts and approaches. Pages 29-42 in G. W. Barrett and R. Rosenberg, editors. Stress effects on natural ecosystems. John Wiley and Sons, New York, New York.

Boesch, D. F., and R. R. 1981. Response to stress in marine benthic communities. Pages 179-200. In: G.W. Barrett and R. Rosenberg (eds.). Stress effects on natural ecosystems. John Wiley and Sons, New York, New York. in G. W. Barrett and R. Ro-

senberg, editors. Stress effects on natural ecosystems. John Wiley and Sons, New York, New York.

Calow, P. 1989. Proximate and ultimate responses to stress in biological systems. Pages 173-181 in P. Calow and R. J. Berry, editors. Evolution, ecology and environmental stress. Academic Press, San Diego, California.

Cooch, E., R. F. Rockwell, and S. Brault. 2001. Retrospective analysis of demographic responses to environmental change: a lesser snow goose example. Ecological Monographs 71:377-400.

Cook, T. D., H. Cooper, D. S. Cordray, H. Hartmann, L. V. Hedges, R. J. Light, T. A. Louis, and F. Mosteller, editors. 1992. Meta-analysis for explanation. Russell Sage Foundation, New York, New York.

Cowan, I. M. 1966. Management, response and variety. Pages 55-65 in F. F. Darling and J. P. Milton, editors. Future Environments of North America. The Natural History Press, Garden City, New York.

Dillon, T. M., and M. P. Lynch. 1981. Physiological responses as determinants of stress in marine and estuarine organisms. Pages 227-242 in G. W. Barrett and R. Rosenberg, editors. Stress effects on natural ecosystems. John Wiley and Sons, New York, New York.

Fore, L. S., J. R. Karr, and R. W. Wisseman. 1996. Assessing invertebrate responses to human activities: Evaluating alternative approaches. Journal of the North American Benthological Society 15:212.

Francoeur, S. N. 2001. Meta-analysis of lotic nutrient amendment experiments: Detecting and quantifying subtle responses. Journal of the North American Benthological Society 20:358.

Frid, A. 2003. Dall's sheep responses to overflights by helicopter and fixed-wing aircraft. Biological Conservation 110:387-399.

Gowan, C., and K. D. Fausch. 1996. Long-term demographic responses of trout populations to habitat manipulation in six Colorado streams. Ecological Applications 6:931.

Hartley, M. J., and M. L. Hunter, Jr. 1998. A meta-analysis of forest cover, edge effects, and artificial nest predation rates. Conservation Biology 12:465-469.

Herbeck, L. A., and D. R. Larsen. 1999. Plethodontid salamander response to silvicultural practices in Missouri Ozark forests. Conservation Biology 13:623-632.

Hertzberg, R. C., and L. K. Teuschler. 2002. Evaluating quantitative formulas for dose-response assessment of chemical mixtures. Environmental Health Perspectives Supplements 110:6.

Hocutt, C. H., R. F. Denoncourt, and J. R. Stauffer, Jr. 1982. Observations of behavioural responses of fish to environmental stress in situ. Journal of Applied Ecology 19:443- 451.

Koehn, R. K., and B. L. Bayne. 1989. Towards a physiological and genetical understanding of the energetics of the stress response. Pages 157-171 in P. Calow and R. J. Berry, editors. Evolution, ecology and environmental stress. Academic Press, San Diego, California.

Leffler, J. W. 1981. Ecosystem responses to stress in aquatic microcosms. Pages 102-119 in J. H. Thorpe and J. W. Gibbons, editors. Energy and environmental stress in aquatic systems. NTIS, Springfield, Virginia.

Levin, L., H. Caswell, T. Bridges, C. Dibacco, D. Cabrera, and G. Plaia. 1996. Demographic responses of estuarine polychaetes to pollutants: Life table response experiments. Ecological Applications 6:1295.

Liess, M. 2002. Population response to toxicants is altered by intraspecific interaction. Environmental Toxicology and Chemistry 21:138.

Liess, M., and R. Schulz. 1999. Linking insecticide Contamination and population response in an agricultural stream. Environmental Toxicology and Chemistry 18:1948.

Maltby, L. 1999. Stress in Ecological Systems - Studying stress: The importance of organism-level responses. Ecological Applications 9:431.

Marschall, E. A., and L. B. Crowder. 1996. Assessing population responses to multiple anthropogenic effects: A case study with brook trout. Ecological Applications 6:152.

Mehlman, D. W. 1997. Change in avian abundance across the geographic range in response to environmental change. Ecological Applications 7:614.

Mooney, H. A., and M. Godron. 1983. Disturbance and ecosystems, components of response. Springer-Verlag, Heidelberg, West Germany.

Myers, R. A., B. R. MacKenzie, K. G. Bowen, and N. J. Barrowman. 2001. What is the carrying capacity for fish in the ocean? A meta-analysis of population dynamics of North Atlantic cod. Canadian Journal of Fisheries and Aquatic Sciences 58:1464-1476.

Sibly, R. M., and P. Calow. 1989. A life-cycle theory of responses to stress. Pages 101-116 in P. Calow and R. J. Berry, editors. Evolution, ecology and environmental stress. Academic Press, San Diego, California.

Stalmaster, M. V., and J. R. Newman. 1978. Behavioral responses of wintering bald eagles to human activity. Journal of Wildlife Management 42:506-513.

Stephens, P. A., and W. J. Sutherland. 1999. Consequences of the Allee effect for behavior, ecology and conservation. Trends in Ecology and Evolution 14:401-405.

Strayer, D. L., S. J. Sprague, and S. Claypool. 1996. A range-wide assessment of populations of *Alasmidonta heterodon*, an endangered freshwater mussel (Bivalvia:Unionidae). Journal of the North American Benthological Society 15:10.

Sumpter, D. J. T., and D. S. Broomhead. 2001. Relating individual behavior to population dynamics. Proceedings of the Royal Society Biological Sciences Series B 268:925-932.

Suter, G., III, T. Vermeire, W. Munns, and J. Sekizawa. 2003. Framework for the integration of health and ecological risk assessment. Human and Ecological Risk Assessment 9:281-301.

Swengel, A. B. 2001. A literature review of insect responses to fire, compared to other conservation managements of open habitat. Biodiversity and Conservation 10:1141-1169.

Van Dyne, G. M. 1981. Response of shortgrass prairie to man-induced stresses as determined from modeling experiments. Pages 57-70 in G. W. Barrett and R. Rosenberg, editors. Stress effects on natural ecosystems. John Wiley and Sons, New York, New York.

Weed, D. L. 2000. Interpreting epidemiological evidence: how meta-analysis and causal inference methods are related. International Journal of Epidemiology 29:387-390.

Winemiller, K. O., and A. A. Anderson. 1997. Response of endangered desert fish populations to a constructed refuge. Restoration Ecology 5:204-213.

Zeise, L., D. Hattis, M. Andersen, A. Bailer, S. Bayard, C. Chen, H. Clewell, R. Conolly, K. Crump, D. Dunson, A. Finkel, L. Haber, A. Jarabek, R. Kodell, D. Krewski, D. Thomas, T. Thorslund, and J. Wassell. 2002. Improving risk assessment: research opportunities in dose response modeling to improve risk assessment. Human and Ecological Risk Assessment 8:1421-1444.